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REVIEW ARTICLE
Tick toxicity in cats caused by *Ixodes* species in Australia: a review of published literature

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Tick toxicity in cats caused by *Ixodes holocyclus* and related species is a common medical condition on the east coast of Australia. Intoxication typically causes a flaccid ascending neuromuscular paralysis and clinical signs can include anxiety, dysphonia, hind limb weakness and/or ataxia, pupillary dilation, respiratory signs and possible bladder voiding dysfunction. Diagnosis is made with a combination of appropriate clinical signs and visualisation of tick(s) on a thorough body search. Cases are classified clinically using a scoring system, which grades neuromuscular weakness and respiratory compromise. The mainstays of treatment are tick removal, administration of tick antitoxin serum and intensive supportive care. Given a prompt and appropriate management regimen, prognosis is good, according to available literature. Most of the literature concerning tick toxicity in cats is anecdotal in nature and an evidence-based review of what is known of this condition has not previously been published.

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Tick toxicity in Australia is usually caused by the hard-bodied tick, *Ixodes holocyclus*, indigenous to the eastern coastline from northern Queensland to Victoria (Ilkiw 1983, Prescott 1984). *Ixodes hirsti* and *Ixodes cornuatus* have also been implicated in feline intoxications (Roberts 1961, Mason et al 1974). Although an estimated 20,000 domestic animals are affected by this tick annually in Australia (Stone 1988), the prevalence of this syndrome in cats is not known. While the clinical signs, pathophysiology and treatment of tick toxicity have been investigated in dogs (Dodd 1921, Clunies Ross 1927, Ilkiw and Turner 1987a,b, Ilkiw et al 1987, 1988, Atwell et al 2001, Campbell and Atwell 2001), similar studies in the cat have not been reported. Table 1 shows the quality of evidence available for this review. The aim of this paper is to review the available published literature concerning *Ixodes* species intoxication in cats using evidence-based criteria to highlight the need for further research, especially in the areas of optimal treatment regimens and clinically useful prognostic indicators.

Epidemiology

Although tick toxicity in domestic animals can be seen all year round in coastal habitats, the number of cases usually peak in spring and summer associated with the predominance of adult female ticks at this time (Doube 1974). The distribution of the paralysis tick on the east coast of mainland Australia mirrors that of the long-nosed bandicoot, an Australian native mammal, which is an important host for the tick (Fig 1). One retrospective study reported that the majority of cats with tick toxicity are presented for veterinary assessment in the spring month of October in south-east Queensland, however, peak numbers are likely to vary with geographic location (Musca and Gunew 2004). Cats are usually presented because the owners have identified a tick or because

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Table 1. Quality of evidence of published literature on feline tick toxicity used for this review

Reference	Peer-reviewed?	Analytical or descriptive	Source of evidence	Number of cases reported
Atwell (2002)	Yes	Descriptive	Case report	1
Atwell and Campbell (2001)	Yes	Analytical	Retrospective survey	Records from 6054 cats
Atwell and Fitzgerald(1994)	No	Descriptive	Expert opinion	NS
Bowman et al (2002)	No	Descriptive	Review	NS
Campbell and Atwell (2002)	No	Descriptive	Conference proceedings	NS
Cooper et al (1976)	No	Descriptive	Conference proceedings	NS
Dodd (1921)	Yes	Descriptive	Review	NS
Fitzgerald (1998)	No	Descriptive	Conference proceedings	NS
Furneaux (1969)	No	Descriptive	Expert opinion	NS
Gibbons (1995)	No	Descriptive	Conference proceedings	NS
Gray et al (1974)	No	Descriptive	Expert opinion	NS
Ilkiw (1983)	No	Descriptive	Review	NS
Jones (1991)	No	Descriptive	Conference proceedings	NS
Kerr (1976)	No	Descriptive	Expert opinion	NS
Knott (1961)	Yes	Descriptive	Review	NS
Malik (1998)	No	Descriptive	Conference proceedings	NS
Malik and Farrow (1991)	Yes	Descriptive	Review	NS
Mason et al (1974)	Yes	Descriptive	Case report	1
Musca and Gunew (2004)	No	Descriptive	Case series	158
Oxley (1981)	No	Descriptive	Expert opinion	NS
Prescott (1984)	Yes	Descriptive	Review	NS
Roberts (1961)	No	Descriptive	Letter	2
Schull (2004)	Yes	Descriptive	Case report	1
Seddon (1968)	No	Descriptive	Review	NS
Seubert (1993)	No	Descriptive	Expert opinion	NS
Strakosch (1988)	No	Descriptive	Expert opinion	NS
Wilkinson (1990)	No	Descriptive	Review	NS
Wilson (1986)	No	Descriptive	Case report	1

NS = not stated.

the cat has developed clinical signs of tick toxicity. There is usually one engorging female tick, but large numbers of larvae or nymphs can also induce paralysis (Jones 1991). Attachment sites are mostly inaccessible to scratching and grooming (Prescott 1984), with the most commonly reported anatomical locations including the head, under the chin, the neck, between the shoulder blades, and caudal to the elbow (Knott 1961, Seddon 1968, Prescott 1984, Atwell 2002, Bowman et al 2002, Schull 2004). Tick attachment sites have also been described on the flanks, tail, external anus, inside the anus, and amongst the transverse palatine ridges of the hard palate (Seddon 1968, Jones 1991). A review of 158 feline cases in south-east Queensland reported that longhaired cats with an outdoor range seemed most at risk and that the average age of cats was 4.7 years (with a range of 10 weeks to 14 years) (Musca and Gunew 2004).

Pathophysiology

While studies investigating the pathophysiology of toxicity in cats have not been reported, experimental studies in mice have confirmed that the class of toxins produced by *I holocyclus* (holocyclotoxins) are active at the neuromuscular

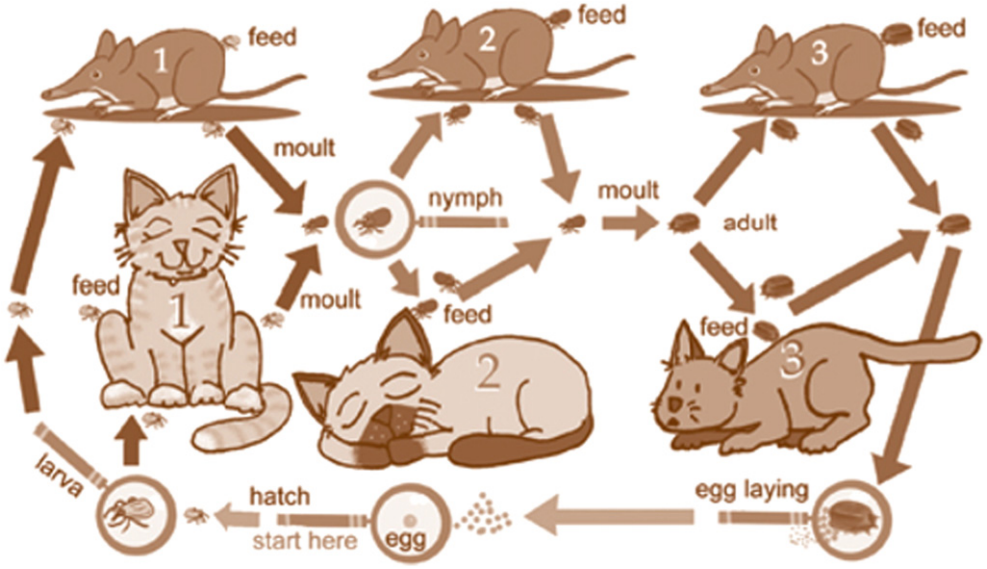

Fig 1. The life cycle of the Australian paralysis tick, *Ixodes holocyclus*, includes three hosts and multiple moult cycles. The long-nosed bandicoot and the cat may each serve as hosts during the life cycle. Musca and Gunew (2004) reproduced with permission from Frank Gaschk (2007).

junction and interfere with the presynaptic release of acetylcholine (Cooper and Spence 1976). The toxin may also interfere with acetylcholine release from parasympathetic nerves, which may account for other clinical manifestations such as pupillary dilation and dysfunction of the larynx and bladder (Cooper et al 1976, Grattan Smith et al 1997, Atwell 2002, Campbell 2002). In the dog, myocardial dysfunction and left-sided congestive heart failure has been noted (Campbell 2002). Localised paralysis at the site of tick attachment has also been reported, with or without systemic clinical signs of clinical intoxication (Anonymous 1999).

Clinical signs

Cats with tick toxicity generally exhibit clinical signs similar to those noted in dogs (Dodd 1921, Seddon 1968, Malik and Farrow 1991), but early clinical changes in cats may be subtle (Cooper et al 1976) such as dysphonia or aphonia due to laryngeal paralysis (Strakosch 1988, Bowman et al 2002), or vague such as an unkempt appearance (Atwell 2002). Hind limb weakness and/or ataxia are the most common presenting signs (Strakosch 1988, Wilkinson 1990, Malik 1998, Musca and Gunew 2004) and withdrawal reflexes weaken as the syndrome advances (Bowman et al 2002) until the cat is unable to rise from lateral recumbency (Musca and Gunew 2004). The tail may remain unaffected by the ascending paralysis in some cases (Musca and Gunew 2004). Pupillary dilation is also a common finding (Mason et al 1974, Cooper et al 1976, Strakosch 1988, Wilkinson 1990, Malik and Farrow 1991).

Respiratory distress has been reported frequently in cats with tick toxicity (Cooper et al 1976, Strakosch 1988, Wilkinson 1990, Malik 1998, Musca and Gunew 2004, Schull 2004) but the degree of respiratory involvement varies from case to case (Strakosch 1988, Seubert 1993). Respiratory signs may appear late in the syndrome (Mason et al 1974), or may be present in the absence of clinically-evident neuromuscular disease (Wilson 1986, Musca and Gunew 2004). Specific respiratory signs reported include coughing (Malik and Farrow 1991), expiratory dyspnoea (thought to be associated with severe bronchospasm) and cyanosis (Wilson 1986, Atwell 2002). An audible expiratory grunt has been noted in some cats with tick toxicity (Malik 1998, Musca and Gunew 2004, Schull 2004). An explanation for this phenomenon was suggested by one investigator following the examination of laryngeal function in a cat with tick intoxication. It was hypothesised that cats with tick toxicity are able to maintain active abduction of the vocal cords during the inspiration, but passive adduction of the vocal cords occurs during expiration, leading to some degree of forced obstructive exhalation with stridor as the vibrating vocal cords are forced apart (Atwell 2002).

Retching, vomiting, or regurgitation may occur with tick toxicity (Malik 1998, Musca and Gunew 2004), placing affected cats at risk of aspiration or asphyxia because of impaired gag reflex associated with pharyngeal paralysis (Campbell and Atwell 2002). Bladder voiding dysfunction in cats has also been noted (Furneaux 1969, Schull 2004).

Cats with tick toxicity often appear distressed or agitated (Malik and Farrow 1991, Malik 1998, Atwell 2002, Bowman et al 2002) because of physical or functional upper respiratory tract obstruction (Campbell and Atwell 2002) and affected cats can deteriorate quickly when handled (Jones 1991, Fitzgerald 1998, Campbell and Atwell 2002). Signs of anxiety range from tail flicking (Musca and Gunew 2004) to a hyperexcitable state, perhaps akin to panic in humans (Strakosch 1988, Gibbons 1995). After neuromuscular paralysis, stress (for example, in association with handling) is thought to be the next major factor contributing to death in affected cats (Jones 1991), as stress-induced hyperexcitability can immediately precede respiratory failure and death (Strakosch 1988, Gibbons 1995).

Diagnosis and case evaluation

The diagnosis of tick toxicity in cats is generally afforded by the detection of adult and/or juvenile ticks, in addition to typical clinical signs. Occasionally, the diagnosis is presumptive based on the identification of a characteristic tick feeding lesion, accompanied by appropriate clinical signs (Bowman et al 2002). Tick feeding lesions are generally characterised by a central crater surrounded by variable localised inflammation, swelling and, sometimes, oedema (Stone 1988).

A clinical scoring system has been used in practise to standardise the assessment of tick toxicity cases and to aid in monitoring treatment and estimating prognosis (Table 2). While the system was developed for dogs, it has also been widely used for cats (Musca and Gunew 2004). The differentiation between weakness scores 1 and 2 can be problematic in cats, as either anxiety or appendicular weakness causes a tendency to crouch sternally and a reluctance to stand (Campbell and Atwell 2002). To differentiate between anxiety and appendicular weakness, some authors have advocated dropping the cat from a height of approximately 30 cm. If the cat lands normally, it is simply anxious, but an abnormal landing (eg, collapse) can be interpreted as appendicular weakness (Fitzgerald 1998, Campbell and Atwell 2002). The use of this technique in the context of this disease could be controversial as it may heighten anxiety in stressed patients.

Table 2. Tick toxicity clinical scoring system (adapted from Atwell et al 2001)

Weakness score	Respiratory score
1 − Mild ataxia or paresis	A − No clinical respiratory compromise
2 − Able to stand unaided but cannot walk	B − Elevated respiratory and heart rate
3 − Unable to stand, can right	C − Restrictive breathing, gagging, retching
4 − Unable to right	D − Expiratory grunt, dyspnoea and cyanosis

Treatment

There are three important components of the treatment of tick toxicity in cats − the removal of adult tick(s) or their juvenile forms, the administration of canine-derived tick antitoxin serum (TAS), and the instigation of supportive care (Bowman et al 2002).

Removal of tick(s)

Multiple thorough searches of the entire body are indicated (Strakosch 1988, Gibbons 1995, Bowman et al 2002), and are facilitated by clipping in longhaired cats to optimise the chances of retrieving the entire tick burden (Bowman et al 2002). One method of tick removal involves the use of opened scissors, which are manoeuvered between the tick and the cat and then used to lever the tick from its attachment site (Malik 1998, Bowman et al 2002). In some cases, if ticks are removed soon after the attachment, toxicity may not develop (Bowman et al 2002).

Administration of TAS

Once clinical signs are apparent, canine-derived TAS is generally administered to neutralise the effects of the tick toxin(s) (Bowman et al 2002), but may be initially withheld from mildly affected cats pending their response to tick removal in hospital (Malik and Farrow 1991). While the slow administration of TAS via the intravenous route in cats is commonly used (Cooper et al 1976, Kerr 1976, Ilkiw 1983, Strakosch 1988, Bowman et al 2002), the intraperitoneal

route (Gray et al 1974, Cooper et al 1976, Smith 1981, Ilkiw 1983, Malik and Farrow 1991) has also been suggested to slow the rate of TAS delivery, thereby reducing the risk of subsequent reactions to the canine-derived product (Campbell and Atwell 2002). In a retrospective review, 61% of 158 cases were given TAS intravenously and 39% via the intraperitoneal route. Based on available case histories, the authors reported that clinical outcomes following both routes were satisfactory, but all cases of TAS reactions (5%, 8/158) occurred in cats that were given TAS via the intravenous route (Musca and Gunew 2004). It is generally recommended that TAS is warmed and/or diluted with saline prior to intravenous administration in cats (Strakosch 1988, Wilkinson 1990, Jones 1991, Gibbons 1995). The optimal TAS dose rate for cats is unknown and/or may vary due to both tick and host factors. In a recent prospective survey, the TAS dose used by clinicians in practise was found to have little effect on patient mortality or recovery time in dogs (Atwell et al 2001). Recommended dose rates range from 1 ml/kg to 3–10 ml/cat (Cooper et al 1976, Strakosch 1988, Wilkinson 1990, Gibbons 1995, Malik 1998, Bowman et al 2002, Musca and Gunew 2004). Although it has previously been recommended that a small amount of TAS is administered at the tick attachment site in cats to neutralise local tick toxin before it enters the circulation (Jones 1991, Malik 1998), in a recent canine prospective survey, this appeared to have no effect on the clinical outcome (Atwell et al 2001).

The administration of TAS in cats carries some risk of a reaction thought to be anaphylactic or anaphylactoid (Cooper et al 1976, Oxley 1981, Prescott 1984, Gibbons 1995, Fitzgerald 1998, Campbell and Atwell 2002) associated with its derivation from canine serum (Malik and Farrow 1991, Bowman et al 2002). These reactions may occur despite pre-treatment with antihistamines (acepromazine 0.05 mg/kg SC), corticosteroids (prednisolone sodium succinate 10 mg/kg SC) and epinephrine (3 ml of 1:10 000 adrenaline SC) (Malik 1998). A retrospective survey reported that reactions to TAS occurred in 6.2% of treated cats. In 63% of the cats that reacted to TAS, the reaction was characterised by bradycardia, reduced heart sounds, pale mucous membranes, weakness and depression. In the remainder (37%), there was tachycardia, injected mucous membranes, anxiety or restlessness, piloerection on the back of the neck, swelling of the lips, cutaneous wheals, erythema, diarrhoea, vomiting, dyspnoea and coughing (Atwell and Campbell 2001). It is recommended that adrenaline and/or soluble corticosteroids are readily available to treat a possible systemic reaction when TAS is administered (Wilkinson 1990, Malik and Farrow 1991, Malik 1998, Bowman et al 2002).

Supportive care

It is particularly important to minimise stress in the feline tick toxicity patient (Gibbons 1995) by limiting interventions, cage rest in a cool, quiet darkened temperature-controlled area, and/or the administration of sedation or anaesthesia (Strakosch 1988, Atwell and Fitzgerald 1994, Fitzgerald 1998, Malik 1998, Campbell and Atwell 2002). If possible, cats should be maintained in sternal recumbency to minimise ventilation/perfusion mismatch. Laterally recumbent cats should be positioned with the head in extension (Malik 1998) and turned periodically to prevent hypostatic congestion and pneumonia (Strakosch 1988, Wilkinson 1990). Oxygen and fluid therapy requirements should be assessed on an individual basis. Some authors have recommended the use of a submaintenance fluid rate for the first 12–24 h (Malik 1998). Maintenance intravenous fluid therapy may be particularly important if paralysis prevents oral administration of fluids (Wilkinson 1990) and fluid resuscitation may be required if there is protracted vomiting or clinical evidence of dehydration (Gibbons 1995). Regular bladder palpation should be performed to monitor for urine production and bladder size. In cats with advanced paresis, urinary catheterisation or manual bladder expression may be necessary to prevent post-paralysis bladder atony, minimise urine scald and aid in the assessment of hydration status (Furneaux 1969, Wilkinson 1990). A thoracic radiographic workup is recommended for cats with respiratory compromise to rule out the possibilities of pulmonary oedema or aspiration pneumonia. Rarely, cats with tick toxicity may need mechanical ventilatory support (Malik 1998). The corneal surfaces should be protected with ophthalmic lubricant if the blink reflex is absent (Wilkinson 1990).

Pathology

A case report describing necropsy findings in a cat that died following *I cornuatus* intoxication reported cyanosis of the tongue and

nose, and local subcutaneous oedema and blood staining at the site of tick attachment. Other gross lesions included endocardial and epicardial haemorrhages, with the endocardial haemorrhages especially severe on the papillary muscles of the left ventricle. Histologically, the subcutaneous tissues at the site of tick attachment were characterised by haemorrhage and oedema, with extensive local infiltration by polymorphonuclear leukocytes, lymphocytes and plasma cells (Mason et al 1974). One other publication suggested that pulmonary oedema rather than inhalation pneumonia was more common in cats that died from tick toxicity (Gibbons 1995).

Prognosis

The mortality rate in cats with tick toxicity is reported to be much lower than that of dogs (Knott 1961, Seddon 1968). In one large case series, only one fatality was recorded from 158 cases of tick toxicity (0.6%) treated over a 5-year period (Musca and Gunew 2004), in contrast with the reported canine mortality rate of 5% (Atwell et al 2001). The period of hospitalisation reported for cats with tick toxicity generally ranges from 0 to 7 days (Strakosch 1988, Musca and Gunew 2004), and reflects the clinical status of the patient on presentation and probably the treatment and management regimen used (Strakosch 1988). Some cats can harbour ticks without showing any clinical evidence of disease (Seddon 1968, Jones 1991), leading to suggestions that some immunity may develop from natural infestations (Seddon 1968).

Prevention/control

Daily inspection for ticks by the owners is an effective way to prevent tick toxicity in cats with an outdoor range (Bowman et al 2002). As clinical toxicity is generally associated with ticks that have been attached to a host for several days (Clunies Ross 1934), removal soon after initial attachment may preclude the development of toxicity. In cats that are confined to a suburban garden, environmental control by clearing vegetation and avoiding mulch can be helpful. There are few products registered for the prevention of *I holocyclus* infestation in cats. Currently, fipronil (applied as a spray every three weeks) and a pyrethrin rinse (used every three days) can be safely used for this purpose in cats.

Conclusions

Tick toxicity associated with *I holocyclus* and related species remains a common cause of morbidity in cats on the east coast of Australia and warrants a high index of suspicion in outdoor cats from endemic areas with acute neuromuscular weakness of the hind limbs. Anxiety, dysphonia, pupillary dilation and expiratory dyspnoea are also common presenting signs. A thorough body search for ticks or their juvenile forms in affected cats is a vital component of diagnostic workup, and regular tick searches by owners are the mainstay of prevention. Treatment regimens are based on tick removal, administration of TAS and careful supportive, and if necessary, intensive care. Given prompt and appropriate case assessment and management, prognosis is good (based on available literature). Most data regarding tick toxicity in cats are based on descriptive reports from non-peer reviewed literature, anecdotal observations and extrapolations from the condition in dogs. Studies are needed to investigate risk factors, common clinical findings, treatment strategies and to further define this complex disease. Studies investigating prognostic indicators would also be clinically useful. Further work may also quantify clinical differences between the condition in cats and dogs, to ensure that optimal species-specific treatment is provided.

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